

## CLINICAL EXPERIENCE WITH RADIOLOGICAL DETERMINATION OF HEART VOLUME

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Since the basic studies of Rohrer (1916) and Kahlstorf (1932), much work on ellipsoid approximation techniques for cardiac volume estimation has been done in Sweden. Publications such as those of Jonsell (1939), Kjellberg, Lönnroth, and Rudhe (1951), and Maurea, Nylin, and Sollberger (1955) bear testimony to the careful and critical way in which the many problems concerned have been tackled. As a result of such work, there has been evolved a method of estimating heart volume from three simple measurements of the cardiovascular outline as seen in standard postero-anterior and lateral radiographs. The validity of the estimates so obtained can best be judged from the post-mortem studies of Lind (1950) and Friedman (1951), who found remarkably close correspondence between actual heart volume (measured by displacement) and radiologically-estimated volume.

Nylin (1955, 1957) used this method when studying changes in heart volume and blood volume, and our own interest in these volume relations during heart failure first led us to use it. This initial experience with the technique encouraged us to extend its use into routine clinical practice and as a result there are available data from 185 patients, almost all with heart disease. Analysis of these data, here presented, provides information about certain factors affecting heart size both in individual cases and in various groups of patients.

### SUBJECTS AND METHODS

Heart volume measurements were made on 185 patients, all admitted to one ward of the Queen Elizabeth Hospital between 1958 and 1961. Two or more estimations were carried out on 31. Of these patients, 101 were male and 84 female. Apart from one child aged 6 years, they were between 17 and 83 years old. The great majority of them were suffering from some form of heart disease as indicated in Table I.

(a) *Radiological Technique.* The method is basically that of Liljestrand *et al.* (1939). Standard chest radiograms were taken at tube-film distances of 2 m. (postero-anterior film) and 1.5 m. (*left lateral film*) with the patient erect: the prone position, for which Larsson and Kjellberg (1948) claim advantages, could not of course be considered for use in this series which included 76 cases of heart failure. No attempt was made to synchronize exposure with a particular phase of the cardiac cycle as this refinement adds so little to the accuracy of the estimate (Kjellberg *et al.*, 1951; Ruosteenoja *et al.*, 1958). The films were taken in full inspiration so that clear cardiac outlines were better seen and so that the radiograms were also suitable for ordinary routine reporting. Delineation of the posterior cardiac border was facilitated by giving the patient a little barium to swallow just before the lateral film was taken.

From the postero-anterior radiogram the following measurements (in cm.) were obtained:

“l”—the “long axis” of the cardiac ellipsoid—measured from the point of junction of the aorta (or superior vena cava) with the right heart border to the left lower pole of the heart.

“b”—the “short axis”—measured from the right cardio-phrenic angle to the junction of the pulmonary conus and the left ventricle (or left atrial appendage).

TABLE I  
DISTRIBUTION OF PATIENTS ACCORDING TO SEX AND ÆTIOLOGY

Ætiology	No. of patients		
	Male	Female	Total
<i>Cardiac disease</i>			
Rheumatic .. ..	27	41	68
Coronary .. ..	39	7	46
Hypertensive .. ..	11	9	20
Thyrotoxic .. ..	2	13	15
Pulmonary .. ..	8	3	11
Congenital .. ..	6	6	12
No cardiac disease .. ..	8	5	13
Total .. ..	101	84	185

Fig. 1A shows these diameters drawn between the conventional points. Adherence to these fixed points has obvious disadvantages, especially where hearts of extraordinary configuration (e.g. with giant left atria), are concerned. However, after trial of several other methods of constructing these axes it was decided to revert to the use of these conventional points because of their great advantage in ensuring comparability of estimates.

From the left lateral radiogram (Fig. 1B), "d"—the diameter of the cardiac shadow, in the horizontal plane, was obtained. It was usually easy to discern the posterior heart border (which may project well behind the barium-filled œsophagus in some cases) but the anterior outline was sometimes difficult to distinguish and the inner border of the sternum had then to be used. Occasionally pleural effusions, severe pulmonary congestion, or other paracardiac shadows obscured the heart's outline, especially in the postero-anterior film, and precluded measurement of heart volume.

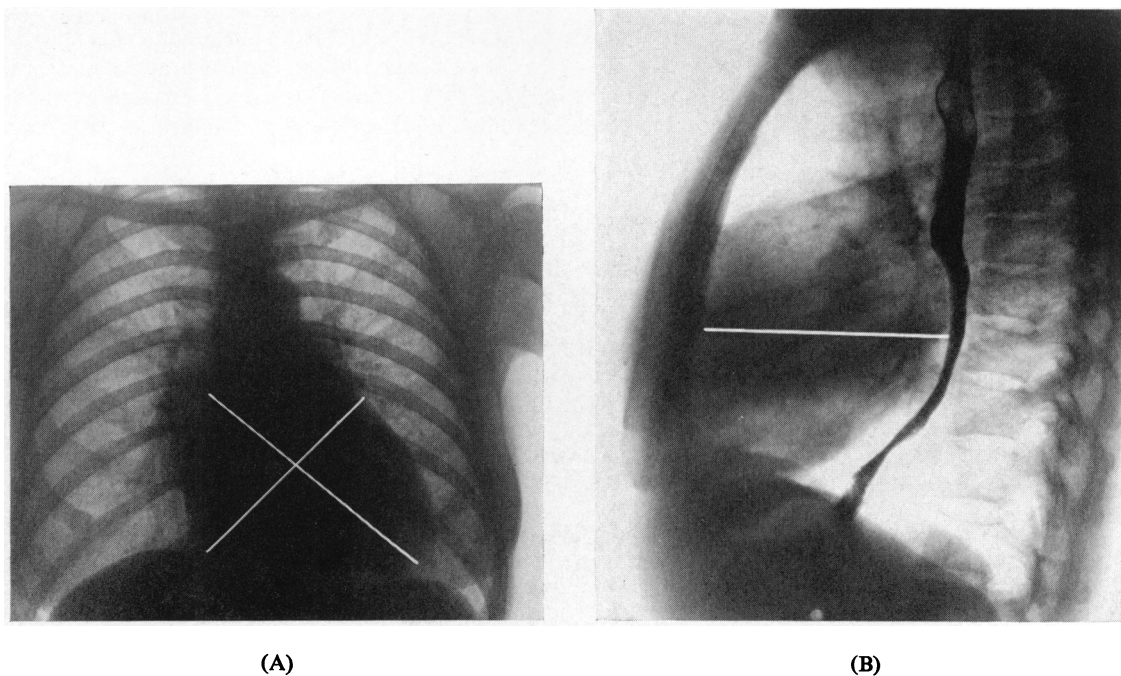


FIG. 1.—(A) Postero-anterior chest radiogram of a case of mitral stenosis showing axes "l" and "b". (B) Left lateral radiograph of the same case showing axis "d".

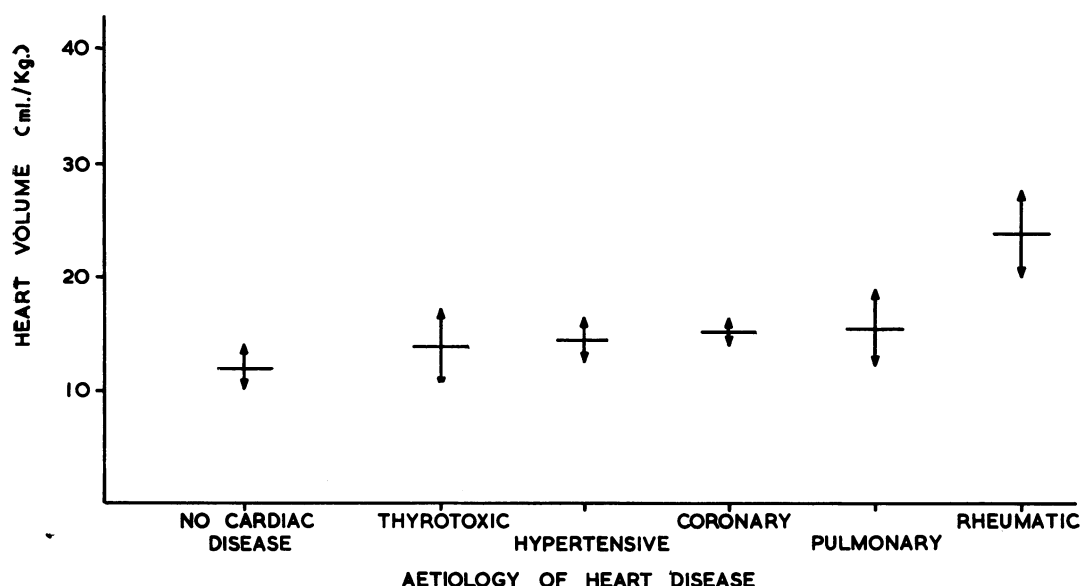


FIG. 2.—Heart volume (mean  $\pm$  2SE) in patients with no history of congestive cardiac failure, grouped according to aetiology.

(b) *The Calculation.* The values for  $l$ ,  $b$ , and  $d$  were substituted in the equation

$$V = K \times l \times b \times d$$

where  $V$  is the cardiac volume in ml. and  $K$  is a factor that includes  $\pi/\epsilon$  and a correction for the x-ray magnification effect.

Throughout this series,  $K$  was separately calculated for each subject in order to take into account the influence of chest thickness upon magnification and also the effect of heart shape and tilting (Larsson and Kjellberg, 1948). It has been found, however, that no worth-while advantage accrues from the use of this refinement (see Appendix) and that, except for hearts of bizarre configuration, it is quite adequate to use a fixed value for  $K$ .

For the tube-film distances specified (2 m. and 1.5 m. for postero-anterior and lateral respectively) and assuming an average chest wall-plate distance of 3.5 cm. for the lateral exposure, and 0.5 cm. for the postero-anterior film,  $K$  will be 0.41 for the great majority of subjects.

## RESULTS

These have been expressed in terms of body weight, i.e. as ml./kg., rather than in relation to height or body surface area as preferred by some (Lysholm, Nylin, and Quarnå, 1934; Maurea *et al.*, 1955), because more data were available for statistical comparison on this basis. The same patterns were shown, but with less sharply defined limits, when the smaller groups for which height measurements were available were compared on a ml./m. or ml./m<sup>2</sup>. basis.

*Influence of Aetiology upon Heart Size.* Fig. 2 shows the mean cardiac volume in ml./kg. body weight in the several groups of cases separated according to the nature of their underlying heart disease: none of these patients had ever, to our knowledge, suffered an attack of congestive failure. Limits for the range of heart volume (on the basis of two standard errors on each side of the mean) are indicated and a group with no heart disease is included for comparison.

Though the mean value in all types of heart disease is above the normal range, only the rheumatic heart disease group stands out clearly from the rest. The other varieties of heart disease here illustrated are not in general associated with gross cardiac enlargement.

*Relation between Heart Size and Atrial Fibrillation.* Fig. 3 shows that, irrespective of the aetiology of the underlying disease, the presence of atrial fibrillation is associated with greater

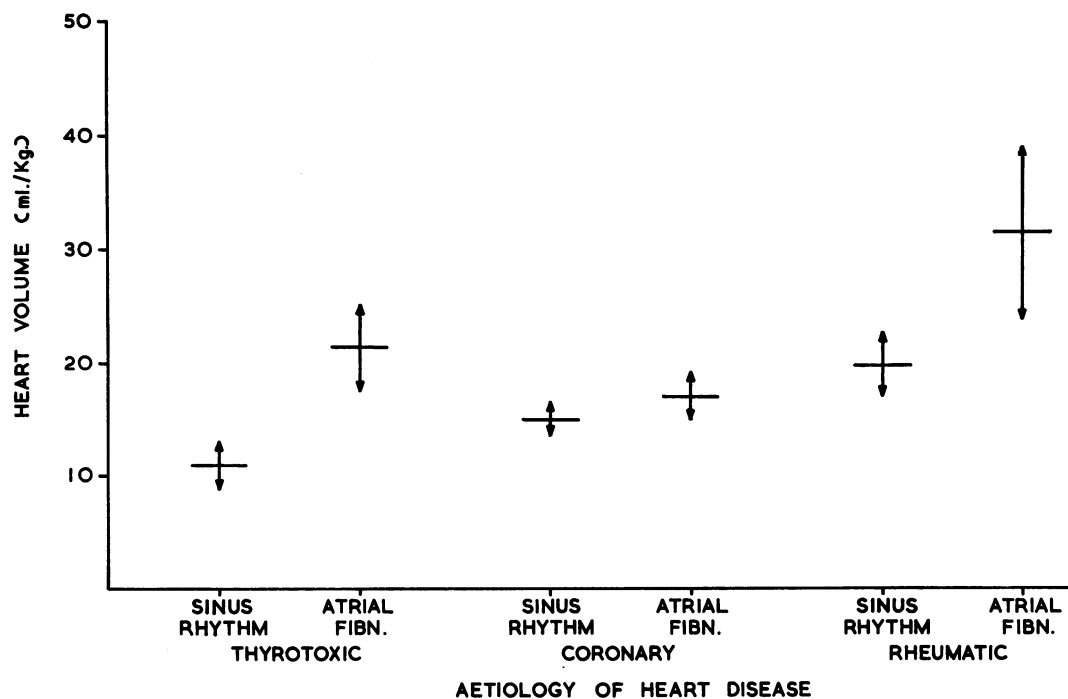


FIG. 3.—Heart volume (mean  $\pm$  2SE) in patients with auricular fibrillation compared with those having similar heart disease but in sinus rhythm. No history of congestive failure.

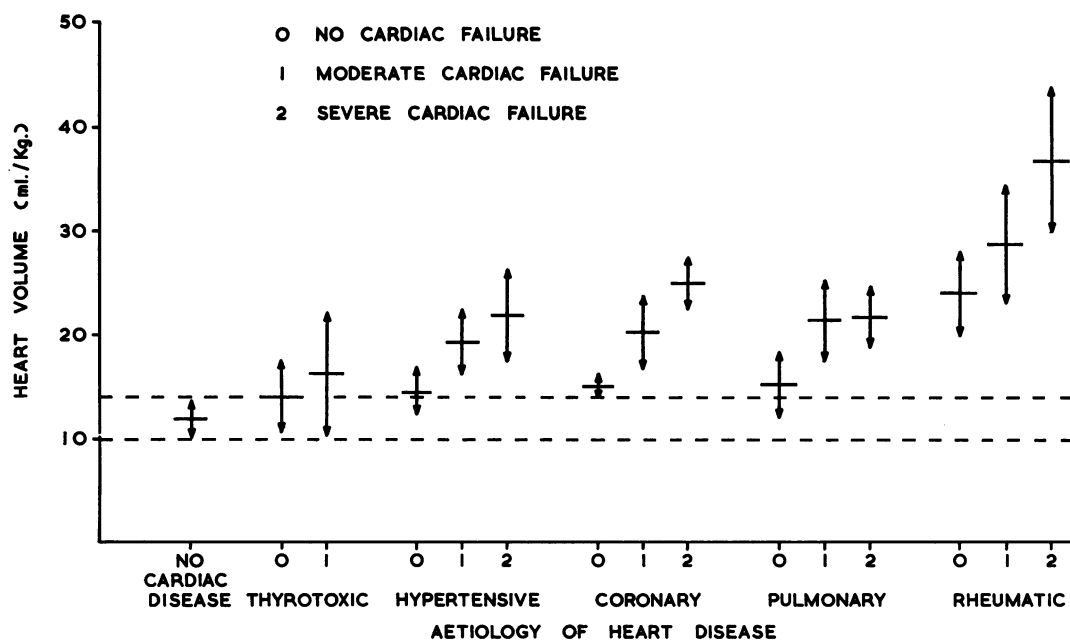


FIG. 4.—Heart volume (mean  $\pm$  2SE) according to aetiology and degree of congestive failure.

cardiac volume than when sinus rhythm persists. Only those groups for whom sufficient data were available for analysis are included. Like the cases used in Fig. 2, none of these had suffered from clinically obvious cardiac failure.

*Effect of Heart Failure.* Fig. 4 shows the progressive increase in heart volume with increasing severity of failure. The latter has been, for this purpose, very simply graded "moderate" or "severe" on the basis of amount of overt oedema. The differences would have been even more striking if heart volume had been expressed in ml./m., so avoiding the masking effect upon heart size expressed in ml./kg. of the concurrent increase in body weight due to the oedema itself.

Fig. 5 shows the influence of a past attack of frank congestive failure on heart size in cases of rheumatic heart disease. As expected, it is seen that the pre-existing cardiac volume is not regained after such an episode, the hearts of these subjects being subsequently somewhat larger than those in cases of similar aetiology who have no history of failure.

Fig. 6 shows the fluctuations in cardiac

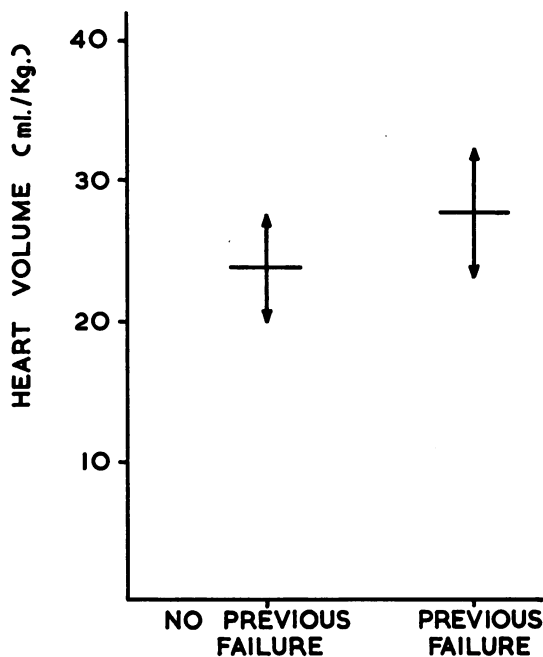


FIG. 5.—Heart volume (mean  $\pm$  2SE) in cases of compensated rheumatic heart disease with and without past history of congestive failure.

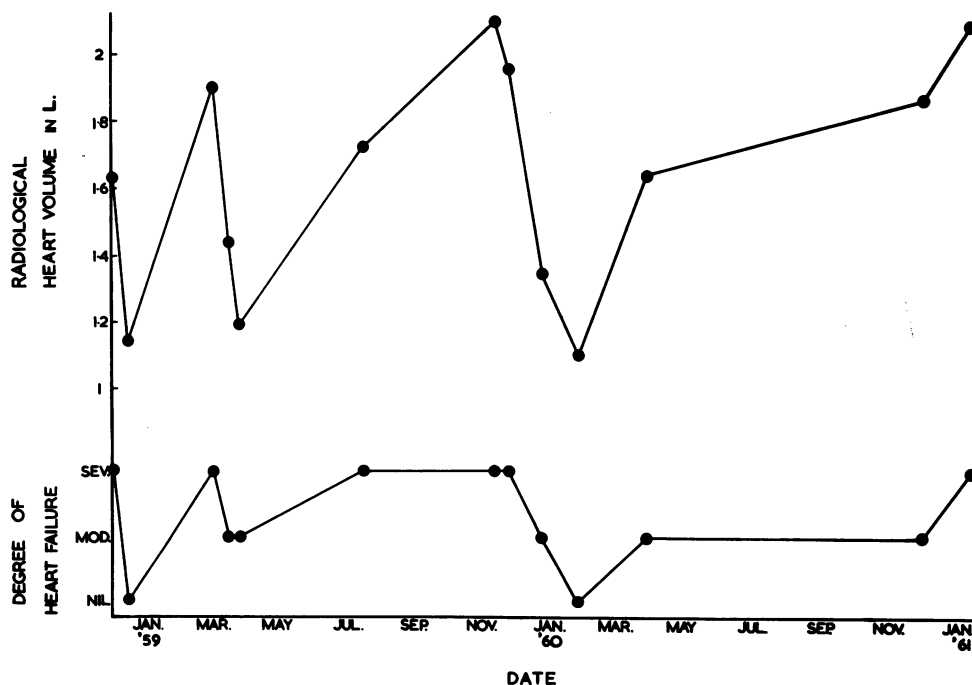


FIG. 6.—Changes in cardiac volume observed in a patient over a period of 27 months and related to improvement and deterioration of congestive failure.

volume in an individual case during treatment of congestive failure. In this figure the actual heart volumes are shown in order to eliminate the effect (on the ml./kg. presentation) of body weight changes due to collection or removal of œdema. The observations were made over a period of 27 months in a 53-year-old man suffering from cor pulmonale.

#### DISCUSSION

Consideration of the apparently very large assumptions involved in ellipsoid-approximation techniques may have led to a natural reluctance to adopt them for clinical use in this country. At first glance it might be thought that volume estimates so obtained must be meaningless as the heart is so obviously not an ellipsoid, it is pulsatile, and often one does not measure diameters quite at right angles to one another in the P-A radiograph. However, in spite of these and other theoretical drawbacks, it has been shown that surprisingly accurate volume measurements can in fact be obtained by these methods, and the work of Friedman (1951) is particularly impressive in demonstrating the close correlation of such estimates, made in cadavers, with true heart volume measurements obtained by displacement. Other studies have shown the relative unimportance of the systolic/diastolic volume difference and of changes in heart size in relation to heart rate (except when very rapid) and phase of respiration. Observer error becomes small after a little practice and should be insignificant where a single observer is regularly employed in making the measurements. The combination of all these variables may account for differences between successive determinations on the same person of up to 12 per cent (Kjellberg *et al.*, 1951). Differences greater than this should always be regarded as indicative of a real change in heart size and much smaller differences will often prove significant where serial measurements are being compared.

For some research purposes this scale of potential error may prove unacceptable and in such circumstances a tomographic method (Thurn, 1959) may provide the more accurate heart volume estimates required. For routine clinical use, however, and in some types of clinical research, the ellipsoid-approximation estimates are sufficiently accurate and afford a ready means of monitoring changes in heart size such as occur during the treatment of congestive cardiac failure. Statement of heart size in terms of volume in this way seems preferable to the use of the transverse cardiac diameter or other measurements which take no account of the depth dimension.

Apart from their use in individual cases as an aid in prognosis and in assessing the efficacy of treatment these volume estimates also allow comparison of heart size on a group basis. From such data it is no surprise to find that atrial fibrillation and congestive failure are associated with increased cardiac volume and hearts that are the seat of chronic rheumatism are bigger than those in the other groups we have studied. It is interesting, however, to see how little difference clinically evident coronary artery disease appears to make to overall heart size (unless failure supervenes) although it must be noted that our small series contained no cases of cardiac aneurysm complicating myocardial infarction. Again, although the number of cases is yet too small for statistical analysis and presentation here, it seems that patients with pure mitral stenosis, not in failure, have much smaller hearts than those with mixed valvular lesions. Wider adoption of these simple volume estimates as the standard means of expressing overall cardiac size would soon decide such points and perhaps uncover additional facts of possible diagnostic importance.

#### SUMMARY

Heart volume estimations have been made on 185 patients by means of a simple radiological technique.

Analysis of these measurements shows an association between heart volume and ætiology of heart disease and illustrates the influence of cardiac failure and atrial fibrillation upon heart size.

Routine radiological heart volume measurement facilitates appraisal of changes in heart size during periods of observation and allows comparison of groups of cases dealt with during day-to-day clinical practice.

We wish to express our thanks to Dr. O. Brenner for permission to study cases under his care and for much valuable criticism and advice. Our thanks are due also to Dr. O. E. Smith and members of the staff of the Department of Radiology, Queen Elizabeth Hospital, to Mr. T. F. Dee for reproduction of the Figures, and to Miss B. Howland for secretarial assistance.

## APPENDIX

*Analysis of Effect of Tilt Factor and Magnification Factor on Measurement of Cardiac Volume*

(a) *Tilt Factor.* In each case an index was calculated from the formula

$$1.2736 \frac{d^2}{lb}$$

where d, l, b refer to the axes defined above in the "methods" section.

This index was then used to derive the "tilt and shape" factor from a modification of the composite graph empirically obtained by Larsson and Kjellberg, (1948).

The tilt factor was calculated for each of the 258 observations made during the course of this study. The results obtained may be summarized as follows:

$$\text{Mean tilt factor} = 1.013 \quad \sigma = 0.0167.$$

This represents an average correction of 1.3 per cent.

The range of the tilt factor was from 1.000 to 1.095. The distribution of the tilt factor is shown in Table II.

If no allowance had been made for the tilt factor, the error involved would have exceeded 5 per cent in only 12 cases (4.6%). It would never have exceeded 10 per cent.

TABLE II

Tilt factor	No. of observations
1.000-1.009	152
1.010-1.019	48
1.020-1.029	24
1.030-1.039	15
1.040-1.049	7
>1.050	12

TABLE III

Magnification factor	No. of observations
<0.3800	0
0.3800-0.3895	2
0.3900-0.3995	13
0.4000-0.4095	84
0.4100-0.4195	105
0.4200-0.4295	43
>0.4300	11

(b) *Magnification Factor.* This was calculated in each of the 258 cases from a nomogram based on the "centre of heart to skin" distances in both projections.  $\pi/\epsilon$  is, for convenience, included in this single factor, hereafter called the magnification factor.

The results may be summarized as follows:

$$\text{Mean magnification factor} = 0.4123; \quad \sigma = 0.0693$$

Range of magnification factor = 0.3810-0.4450.

Distribution of magnification factor according to range is shown in Table III.

If a value of 0.41 had been used as a constant magnification factor the percentage error would have been greater than 5 per cent in only 6 cases (2.3%). It would never have exceeded 9 per cent.

This magnification factor is, of course, valid only for films obtained by the specified radiographic technique.

## REFERENCES

- Friedman, C. E. (1951). Heart volume, myocardial volume and total capacity of the heart cavities in certain chronic heart diseases; a clinic, roentgenologic and patho-anatomic investigation of the problem of cardiac hypertrophy and dilatation and amount of residual blood of the heart. *Acta med. scand.*, **140**, suppl. 257.
- Jonsell, S. (1939). A method for the determination of the heart size by teleroentgenography. *Acta radiol. (Stockh.)*, **20**, 325.
- Kahlstorf, A. (1932). Über eine orthodiographische Herzvolumenbestimmung. *Fortschr. Röntgenstr.*, **45**, 123.
- Kjellberg, S. R., Lönroth, H., and Rudhe, U. (1951). The effect of various factors on the roentgenological determination of the cardiac volume. *Acta radiol. (Stockh.)*, **35**, 413.
- Larsson, H., and Kjellberg, S. R. (1948). Roentgenological heart volume determination with special regard to pulse rate and the position of the body. *Acta radiol. (Stockh.)*, **29**, 159.
- Liljestrand, G., Lysholm, E., Nylin, G., and Zachrisson, C. G. (1939). Normal heart volume in man. *Amer. Heart J.*, **17**, 406.

- Lind, J. (1950). Heart volume in normal infants; a roentgenological study. *Acta radiol. (Stockh.)*, suppl. 82.
- Lysholm, E., Nylin, G., and Quarnå, K. (1934). The relation between the heart volume and stroke volume under physiological and pathological conditions. *Acta radiol. (Stockh.)*, **15**, 237.
- Maurea, Nylin, G., and Sollberger, A. (1955). Normal heart volume. *Acta cardiol. (Brux.)*, **10**, 336.
- Nylin, G. (1955). Blood volume and residual volume of the heart in decompensation. *Amer. Heart J.*, **49**, 803.
- (1957). The clinical applicability of roentgenological heart volume; determination with special reference to the residual blood. *Acta cardiol. (Brux.)*, **12**, 588.
- Rohrer, F. (1916). Volumbestimmung von Körperhöhlen und Organen auf orthodiagraphischem Wege. *Fortschr. Röntgenstr.*, **24**, 285.
- Ruosteenoja, R., Linko, E., Lind, J., and Sollberger, A. (1958). Heart volume changes at rest and during exercise. *Acta med. scand.*, **162**, 263.
- Thurn, P. (1959). Zur röntgenologischen Volumenmessung des Herzens. *Fortschr. Röntgenstr.*, **90**, 290.